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Chapter 10: Burn Wound Management

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INTRODUCTION

CHAPTER OBJECTIVES

At the end of this chapter, the learner will be able to:

1. **Classify burn wounds according to the depth of tissue injury using the American Burn Association terminology.**
2. **Estimate the percentage of total body surface area of a burn using the "Rule of Nines."**
3. **Appreciate the different mechanisms of burn injuries.**
4. **Recognize the critical aspects of the initial evaluation and assessment of a burn injury.**
5. **Estimate the fluid and caloric requirements of a burn patient.**
6. **Understand the different nonsurgical and surgical treatment modalities for burns and identify when surgical treatment is required.**
7. **Distinguish the discrete complications that can occur after a burn injury and discuss how they are managed.**
8. **Address the major physical and psychological objectives of rehabilitation in a burn patient.**

Epidemiology

The World Health Organization estimates that almost 200,000 deaths annually are attributed to burns, and the vast majority of burn morbidity worldwide is related to nonfatal burn injuries.[1] In the United States alone over 45,000 patients each year require hospitalization for burns,[2] creating a significant burden for the health care system. Over 90% of the burn injuries are deemed preventable occurrences. Mortality has significantly decreased with the development of regional burn units, establishment of multidisciplinary treatment teams, and improved critical care strategies focused on optimizing resuscitation, early surgical interventions, infection control, and nutrition. The American Burn Association reports the current survival rates are greater than 96%.[2] With increasing survival of this patient population, greater emphasis has been placed on long-term rehabilitation geared at restoration of function and activities of daily living, correction of esthetic deformities, and improvement of the psychosocial well-being. With this paradigm shift in burn care toward emphasis on optimizing post-burn quality of life, newer interventions are being developed and applied even during the acute phase of burn care to facilitate such improvements for burn survivors.

Pathophysiology

The term *burn injury* is often employed as an umbrella term that encompasses a vast array of unique mechanistic etiologies which eventually result in injury to skin and underlying structures. These mechanisms are classically separated into four main categories: thermal, chemical, radiation, and electrical. Thermal etiologies include flame, contact, scald, and frostbite. Thermal injury to the skin is the result of the direct energy transfer to the tissue in relation to temperature and contact time. Temperature is in actuality a derivative of the average kinetic energy of the molecules within a system, or in the case of burn injury, a substance (ie, boiling water), such that the temperature of any substance represents a potentially transferable

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molecular kinetic energy (KE_{avg} = 3/2 κT; κ = Boltzmann's constant, T = Kelvin) to some other substance (ie, skin). Transfer of this stored kinetic energy to cellular structures of the skin results in denaturation of proteins, vaporization of water, and thrombosis of cutaneous blood vessels, thus resulting in tissue and cell death. The rapid coagulation of protein occurs in the setting of irreversible thermal cross-linking that may limit depth progression when compared to other burn mechanisms such as that seen with chemical and radiation. This process may be immediate in the case of high temperature and/or prolonged contact time, but may also be potentiated by the patient's premorbid condition, injury status, and local inflammatory factors. The delineation between hot and cold thermal mechanisms (ie, flame, contact, scald) strictly translates to the interface through which the stored kinetic energy is transferred and has implications with respect to the contact time required to facilitate such a transfer. For example, contact burns occur when a solid substance (ie, hot iron) contacts skin; a burn injury of a specific depth will occur at a much shorter exposure interval than to that of a liquid or gas of the same temperature. This is because the solid substance has a significantly greater molecular energy density, which allows it to transfer a greater magnitude of energy across the contact interface in a short period of time in comparison to less energetically dense liquid and gas. This means that in order for liquid and gas to achieve the same depth burn as that of a contact mechanism, a higher temperature or longer contact time would be required. This nuance is important to keep in mind when comparing the burn mechanism to the pattern of a burn injury. For example, a mixed mechanism such as hot noodles (boiling liquid and hot solid) will often result in variable burn depths despite both components of the substance having the same “temperature.” This delineation also plays an important role when examining burn patterns and comparing observations to the reported mechanism for investigations related to burn abuse and neglect. Other mechanisms of burn injury related to chemicals, electricity, and radiation will be discussed in further detail later in the chapter as tissue damage in these instances is not always a direct result of kinetic energy but rather more complex biochemical and electrophysical interactions.

Zones of Injury

In 1953, Douglas M. Jackson published a classic article in the *British Journal of Surgery* describing three histological zones of dermal burn injury. These “zones of injury” have been adapted by burn literature and are still applicable (**FIGURE 10-1**).[3] Zone 1 is the *Zone of Coagulation* and refers to the area of burned tissue that is irreversibly damaged and is no longer viable. Depending on burn size and depth, this area may be addressed with immediate mechanical or subsequent operative debridement. Zone 2 is the *Zone of Stasis* which refers to the area of burned tissue that is potentially reversible if appropriate and expedient post-burn interventions are administered. In particular, adequate intravascular resuscitation, infection prevention, early removal of necrotic or non-viable tissues, nutrition optimization, and restoration of physiologic core body temperature are all components of the treatment algorithm that can minimize conversion of Zone 2 to irreversible Zone 1 injury. Zone 3 is the *Zone of Hyperemia*, which is the area of “unburned” tissue surrounding Zone 2 that is exposed to the nearby release of inflammatory cytokines (eg, interleukins, prostaglandins, bradykinin, substance P, histamine) and subsequently develops vasodilation and increased capillary permeability leading to “hyperemia.” As there is no direct tissue injury, Zone 3 is completely reversible unless infection of adjacent Zone 2 burn results in inflammatory sequelae of the Zone 3 tissue.

FIGURE 10-1

Jackson's concentric zones of burn tissue Jackson's burn theory classifies three concentric zones in relation to the potential viability of the tissue. These regions from the center of the wound to the periphery were labeled as the zone of coagulation, zone of stasis, and the zone of hyperemia. The center zone of coagulation is the area of maximum contact to the thermal source. Tissues in this area undergo a coagulation necrosis as proteins of the extracellular matrix are denatured and vascularity is impaired. Cells will not recover and emphasis is placed on early debridement and prevention of infection. The intermediate zone of stasis is characterized by hypoperfusion and hypometabolism as the numbers of viable cells are substantially reduced. Phenotypically this area appears as blanching erythema when pressure is applied. There is a risk of tissue progression to necrosis if proper care is not taken to preserve tissue perfusion by adequate fluid resuscitation, avoidance of vasoconstrictors, and prevention of infection. Patients with significant comorbidities that impair blood flow such as diabetes, peripheral vascular disease, and tobacco use are at increased risk for irreversible injury. The outer zone of hyperemia also appears as erythema as the result of local vasodilatation. Cells in this zone are completely viable and will recover if protected from further trauma and/or infection.

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BURN WOUND CLASSIFICATION

Depth of Penetration

The severity of the burn injury and treatment algorithm is determined by the depth of penetration and the surface area of injured skin in relationship to the total body surface area (TBSA) of the patient. The previous designation of first-, second-, and third-degree burns has been replaced by the designations of superficial, partial thickness, and full thickness, respectively, because of the heterogeneity within the spectrum of burn depth. While researchers and clinicians have transitioned to the aforementioned depth descriptors, medical coding still utilizes the degree system, so it is important for practitioners treating burn wounds to understand both systems and the translation between the two.

Superficial injuries are burns limited to the epidermis without disruption of epithelial integrity (**FIGURE 10-2**). This injury was previously designated *first-degree burn.* These injuries are characterized by erythema of the skin secondary to vasodilatation of local capillaries. Sunburns are examples of superficial burns and after 3–4 days, the damaged epithelium desquamates and is replaced by regenerating keratinocytes.[4] Importantly, superficial or first degree burns are not included in TBSA calculations as this injury does not violate the dermis and therefore the physiologic derangements from such burns are inconsequential during the treatment of deeper burns. Often, referring providers will incorrectly include superficial or first-degree burns in TBSA calculations, which leads to burn size overestimation, excessive or unnecessary treatments, and inappropriate initiation of Parkland resuscitation.

FIGURE 10-2

Superficial burn Superficial burns involve only the epidermis and will heal in 3–4 days without scarring or medical intervention. Sunburn on the shoulder of this toddler is an example of a superficial burn.

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Partial-thickness, previously *second-degree*, burns are heterogeneous in nature due to the differences in dermal thickness regionally, and in pathophysiology related to differences in superficial and deep injuries to the dermis. The primary state of a partial-thickness burn is that dermal structures are still intact and thus spontaneous healing may occur through migration of epithelial progenitor cells from dermal appendages. However, the healing potential of superficial partial-thickness burn may be much better than a deep partial-thickness burn because of the absolute amount of dermis that is viable. This heterogeneity difference in healing potential and the correlating need for possible excision and grafting have resulted in the designation of superficial partial-thickness *and* deep partial-thickness injury.

Superficial partial-thickness burns are dermal injuries that extend into the papillary dermis (**FIGURE 10-3**). These wounds are sensate to nociceptive, proprioceptive, and light touch stimuli, blanch with pressure, and typically result in blistering as a result of the local inflammatory process between the dermis and the epidermis. In particular, blistering in superficial partial-thickness burns is the result of exudative accumulation at the site of the damaged epidermal–dermal interface and these blisters often remain intact at time of presentation. The blistering is often serous in nature; thus, a hemorrhagic appearance often correlates with deeper partial-thickness burns. These wounds heal within 2–3 weeks by re-epithelialization from retained dermal appendages (i.e. bulge stem cells from base of hair follicles) and rarely form hypertrophic scarring and/or wound contracture; therefore, they do not require excision and grafting.

FIGURE 10-3

A. Superficial partial-thickness burns Superficial partial-thickness burns involve the epidermis and the superficial dermis. They are characterized by intense pain, blanching with pressure, and blistering as a result of the local inflammatory process between the dermis and epidermis. These wounds heal within 2–3 weeks by epithelization and rarely form hypertrophic scarring and/or wound contracture. A scald burn on the lower extremity with sloughing of the epidermis is an example of a superficial partial-thickness burn; the areas that are mottled and nonblanching are deep partial-thickness burns. **B. Superficial partial-thickness burn re-epithelializing** The thermal burn on the plantar foot is superficial partial thickness, and islands of new epithelium can be observed in the middle of the wound. The blistered tissue was debrided, but the wound healed without grafting.

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Deep partial-thickness burns extend into the reticular dermis and are nociceptively insensate, have a mottled white appearance, and do not blanch with pressure as the result of impaired vascularity and capillary refill (**FIGURE 10-4**). Because the free nerve endings responsible for nociception are more superficial within the dermis compared to the other somatosensory nerve receptors contained within skin, often deep partial burns will remain sensate to light touch (ie, Merkel's cells, Meissner's corpuscles), vibration and rapid pressure (Pacinian corpuscle), and stretch and sustained pressure (ie, Ruffini's corpuscles) even when no longer sensitive to pain. This delineation of the type of sensation that is lost within a burn wound helps further clarify the depth of burn when a mixed clinical exam is present. Furthermore, deep partial burns may also be pink rather than white but blanching will be diminished compared to other more superficial areas and there is often a transition point rather than clear demarcation. The reticular dermis contains skin appendages, and injury in this area can cause permanent damage to hair follicles and sebaceous glands, two critical sources of epidermal stem cells required for re-epithelialization. Furthermore, the reticular dermis possesses a distinct lineage of fibroblasts compared to those of the papillary dermis that are specifically responsible for deposition of new extracellular matrix proteins. Deep partial-thickness burns, therefore, damage this fibroblast population that is crucial for wound healing. As a result of damage to stem cell-containing hair follicles and sebaceous glands as well as loss of reticular fibroblasts, deep partial-thickness burns heal in 3–9 weeks by contraction rather than re-epithelialization, which results in significant hypertrophic scarring and wound contracture. Because of the sequelae of delayed healing in deep partial-thickness burns that are allowed to heal by contracture—causing hypertrophic scarring and contractures with significant cosmetic and functional impairments—they are best treated by debridement of necrotic tissue and autologous skin grafting.

FIGURE 10-4

Deep partial-thickness burn Deep partial-thickness injuries extend into the reticular dermis and are insensate, have a mottled white appearance, and do not blanch with pressure as the result of impaired vascularity and capillary refill. Deep partial-thickness burns heal in 3–9 weeks, sometimes with significant hypertrophic scarring and wound contracture; therefore, they are best treated by debridement of necrotic tissue and skin grafts.

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Full-thickness burns are cutaneous injuries extending through the entire dermis and across the subdermal plexus, owing to complete loss of skin's barrier function (**FIGURE 10-5**). The previous designation was a *third-degree burn.* This designation also implies that there is no possibility of healing from the wound base, as all dermal regenerative cells have been obliterated. The physical appearance is often leathery brown or black eschar with no capillary refill, although pink and white colors may be present. The key delineation is that pigmentation is fixed in full thickness injuries because no blanching is present with coagulation of the subdermal plexus. Furthermore, full thickness burn tissue is dead and therefore shrinks or contracts as opposed to partial-thickness tissue which is damaged, not dead, and therefore swells. The saying “dead tissue shrinks, damaged tissue swells” is a helpful one to remember when assessing burn depth. The wounds are insensate with respect to all forms of somatosensation and treatment is focused on early excision and grafting to prevent infection, hypertrophic scarring, and wound contracture. Depending on size and location, most full-thickness injuries are treated with excision and grafting procedures, although excision and delayed primary closure or more complex reconstructive options such as local or free vascularized tissue transfer may be employed when critical structures are exposed or ideal graft-receptive wound beds are not available.

FIGURE 10-5

Full-thickness burn Full-thickness burns extend through the entire dermis and into the subcutaneous tissue. The appearance is leathery brown or black eschar with no capillary refill. The wounds are insensate and treatment is focused on early excision and grafting to prevent infection, hypertrophic scarring, and wound contracture. This foot with extensive eschar and exposed subcutaneous tissue is an example of a full-thickness burn.

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The extent of burn penetration may not be readily apparent at the time of initial evaluation because the burn eschar often impairs delineation of true depth. Although many tools have been used as predictive indicators of depth of injury (eg, biopsy, ultrasound, infrared, or perfusion techniques), none has proven to be as reliable as a physical examination at 48–72 hours postinjury.[5] The initial non-surgical debridement of burn wounds with mechanical methods such as rigorous scrubbing to remove blisters, exudate, coagulum, and eschar is crucial to the accurate assessment of the burn depth.

Size of Burn Injury

An estimate of the burn wound surface area in proportion to the TBSA is used to estimate the total fluid and caloric requirements and is a predictor of morbidity and mortality. For an adult patient, the burn area is often generalized utilizing the “Rule of Nines” for rapidity and ease of assessment (**FIGURE 10-6**). When assessing burn size for patients with BMIs greater than 35, adaptation of the Rule of Nines to the Rule of Sevens is indicated to reduce inaccuracies that occur when generalizing the Rule of Nines to obese patients. In particular, the anterior and posterior torsos represent 28% TBSA, respectively, whereas the lower extremities are 14% and the upper extremities 7%. While the Rule of Nines and Sevens are useful for larger, contiguous burns, the Palmar Surface Area (PSA) technique which equates the patient's own palm and fingers to 1% TBSA is best used for smaller and scattered patterns. It is important to note that age, gender, and BMI all impact PSA relative to TBSA, and can result in twofold errors in estimation when not accounted for by the practitioner. More sophisticated charts such as the Lund–Browder chart are available for more accurately estimating the body surface in relationship to age and may be more useful particularly in the pediatric population (**TABLE 10-1**). The Lund–Browder chart, although more accurate, is more time-consuming in application and is often not employed by non-burn and pre-hospital providers. In order to ameliorate the significant errors that may occur in TBSA estimation, computer- and mobile app–based methods such as Burn Case 3D[6] have been devised. The depths of penetration as well as the TBSA burn are major determinants by the American Burn Association for referral to a regional burn center for a higher level of care (**TABLE 10-2**).

FIGURE 10-6

Rule of Nines The Rule of Nines is used to determine total body surface area that is burned and estimates that each upper extremity accounts for 9% of the total burn surface area or TBSA, and each lower extremity, 18%. In addition, the anterior and posterior trunk is predicted to be 18%, the head and neck 18%, and the perineum 1%. For burns spanning anatomical regions, the volar surface area of the hand may be considered 1% of the TBSA.

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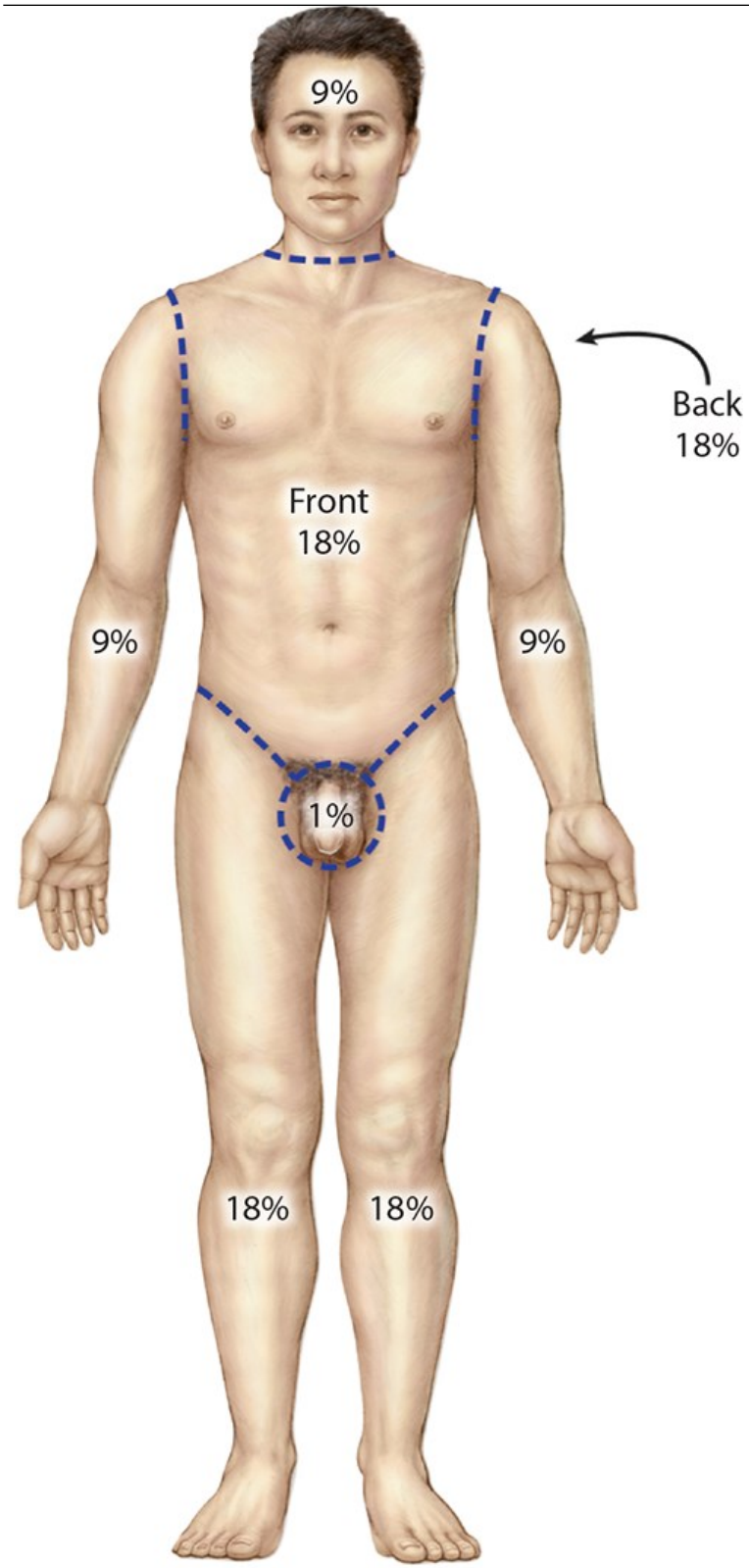

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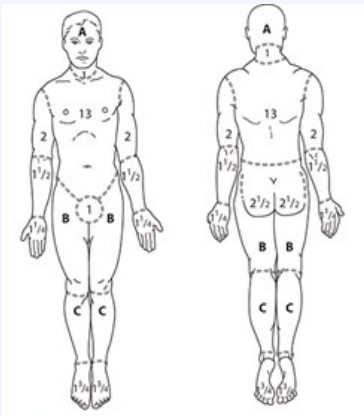
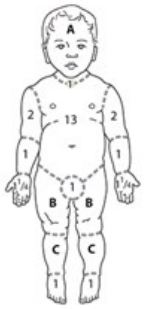
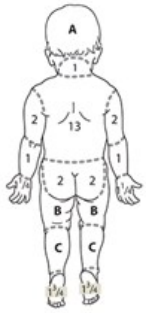
TABLE 10-1

Estimating Total Surface Burn Area







Relative Percentages of Areas Affected by Growth

Area	Age 10	Age 15	Age Adult
A = half of head	5 1/2	4 1/2	3 1/2
B = half of one thigh	4 3/4	4 1/2	4 3/4
C = half of one leg	3	3 1/4	3 1/2

Relative Percentages of Areas Affected by Growth

Area	Age 0	Age 1	Age 5
A = half of head	9 1/2	8 1/2	6 1/2
B = half of one thigh	2 3/4	3 1/4	4
C = half of one leg	2 1/2	2 1/2	2 3/4

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TABLE 10-2

Criteria for Referral to a Regional Burn Center

American Burn Association Criteria for Referral to Regional Burn Unit

- Partial-thickness burns that encompass greater than 10% TBSA
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints
- Third-degree burns in any age group
- Electrical burns, including lightening injury
- Chemical burns
- Inhalation injury
- Burn injury in patients with pre-existing medical conditions that can complicate management, prolong recovery, or affect mortality
- Any patients with burns and concomitant trauma, in which burn injury poses the greatest risk of morbidity and mortality
- Burn children in hospitals without qualified personnel or equipment for the care for children
- Burn injury in patients who will require special social, emotional, or rehabilitative intervention

Data from www.ameriburn.org.

PROGNOSIS

When a patient first enters a burn center and the initial burn evaluation is completed, prognostication is performed to facilitate a better understanding of injury severity as well as discussions with the patient and family members regarding overall prognosis. Initially, when the Parisian surgeon Serge Baux first devised his mortality scoring system in 1961, this calculated score (ie, Baux Score = patient age + % TBSA) translated directly to the patient's mortality rate. In other words, a 60-year-old patient with a 40% TBSA burn had a 100% mortality rate. However, due to significant advances in burn

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critical care and surgical management, this score is now used as a marker of injury severity but not as a direct measure of mortality risk. Recently, to account for the influence of inhalation injury on mortality in burn injury, the Revised Baux Score (rBaux) was formulated by Osler et al. in 2010.[7] This method calculates a score by summating a patient's age, % TBSA, and adding an additional 17 points in the presence of any inhalation injury. For example, a 50-year-old patient with a 30% TBSA burn and inhalation injury would have a total Revised Baux Score of 97 (50 + 30 + 17). With the rBaux, a conversion nomogram may be used to determine predicted mortality. While the rBaux provides a relatively accurate estimate of predicted mortality after severe burn injury, it does not account for other clinical indicators of prognosis such as comorbidities or concomitant trauma.

MECHANISMS OF BURN INJURIES

Scald

Scald burns are the result of contact with hot liquids and are the most common burn etiology in developed countries. The extent of injury is dependent on the temperature and viscosity of the liquid and the duration of exposure, in addition to patient factors and anatomical location. Exposure to hot water above 60°C for 3 seconds can result in deep partial-thickness or full-thickness injury. These injuries typically appear less severe in exposed areas of the body. Clothed areas typically have more extensive injuries because clothing absorbs the heated liquid, prevents evaporative heat dissipation, and maintains contact with the skin for a longer duration of time. In the pediatric population, immersion scald burns with a symmetric distribution and linear demarcations of the upper extremity or lower extremity and/or crease-sparing of perineal and buttocks should warrant suspicion of abuse, and health care professionals are required to notify appropriate child protective agencies (**FIGURE 10-7**). While not within the scope of this chapter, a detailed discussion on the evaluation and management of suspected abuse and negligent burns may be found in “Negligent and Inflicted Burns in Children” by Collier et al. in the July 2017 issue of *Clinics in Plastic Surgery*.[8]

FIGURE 10-7

Scalding burn This toddler's hand is an example of a superficial partial-thickness burn due to scalding water. The depth of tissue injury depends on the liquid temperature and time of exposure. Any suspicion of child abuse with this type of burn should be reported to the Children's Protective Services.

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Scald burns from grease may result in more severe injuries as grease is more viscous than water, therefore resulting in a longer duration of contact from decreased evaporative losses and difficulty removing the offending agent. Although grease can reach higher temperatures (usually around 200°C) compared to water, water has a significantly higher heat capacitance and therefore contains more stored thermal energy for a given temperature. As a result, water can transfer greater energy in a given period of time and cause greater damage per second of exposure. The combination of boiling water and oil results in the greatest degree of damage compared to either in isolation as the oil prevents evaporation of water which facilitates prolonged delivery of stored thermal energy in water to the contacted tissues. These burns therefore frequently result in deep partial-thickness to full-thickness injuries.

Tar is a unique type of scald burn that requires specialized management because it becomes adherent to the skin as it cools and creates a secondary alkaline chemical injury. Treatment is initially focused on rapidly cooling the tar with ice packs to reduce continued thermal injury followed by the slow removal of the tar with petroleum-based ointment such as Neosporin (Johnson & Johnson, New Brunswick, NJ) and Medi-sol (Medi-sol, Oklahoma City, OK) or simple mineral oil. The ointment or oil may need to be applied multiple times in order to totally dissolve the tar. Rapid intervention is warranted even after cooling measures have stopped thermal-based injury, as the alkaline composition of tar can continue to cause underlying liquefactive necrosis. Once dissolved, appropriate assessment can be made regarding the depth of penetration and extent of injury.

Flame

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Flame injuries are the second most common mechanism of burns and the most common mechanism requiring hospitalization. Flame injuries account for 44% of burn admissions in the United States annually.[2] The incidence of flame burns has significantly decreased with the implementation of prevention programs and improved detection methods such as smoke detectors and fire alarms. The major determinant of morbidity and mortality in this patient population is the presence of concomitant inhalation injury.[9] Inhalation injury should be suspected if the burn occurred within an enclosed space or if physical examination reveals singed nasal hair, voice changes (eg, coarse or hoarseness), inspiratory stridor, and/or carbonaceous sputum.

Electrical

Electrical burns have been described as the most devastating burn injuries because they not only involve the skin, but underlying organ systems as well (eg, the cardiovascular and nervous systems). Electrical burn pathophysiology, like that of chemical injury, has a mixed contribution from direct and indirect mechanisms to create tissue injury to both cutaneous and deeper structures. Electrical current is directly damaging to cell membranes as the flow of electrons will generate heat as they flow through relative “high resistance” of these cellular structures. This mechanism creates the entry and exit wounds often seen in electrical injury. As electrical energy travels through the body, it is converted to heat, and the severity of injury is dependent on voltage, magnitude, and type of current (alternating vs direct), the pathway traveled through the victim's body, and the electrical resistance of tissues through which the electricity passes [least-to-most resistance: blood vessels, nerves, muscle, skin (wet < dry), tendon, fat, and bone]. Bone has a relatively high resistance compared to other tissues and heats up with conduction of electrical current such that thermal injury to adjacent muscle can be severe and lead to occult compartment syndrome which is hard to recognize and can be fatal if missed.

Electrical injuries are classified as low voltage (<1000 V) and high voltage (≥1000 V). Low voltage typically involves tissues immediately surrounding the point of initial contact, while high voltage usually results in penetration to deeper organ systems. High-voltage injuries, especially from alternating current sources, create significant tetany that may result in skeletal fracture, even of the spine, and the sustained high-intensity contractions can lead to myocyte necrosis. This muscle damage causes myoglobinuria that can result in renal failure and hyperkalemia, which in turn may lead to life-threatening arrhythmias. Alternating current causes tetanic contractions that can prevent the victim from releasing the source, called the *no-let-go-phenomenon*, thereby resulting in prolonged contact with the electricity and thus more devastating injuries. Smaller body parts such as the hands, fingers, and feet generate more heat because they dissipate less heat to the surrounding tissues, and in many cases injuries to these structures result in complete necrosis (**FIGURE 10-8**).

FIGURE 10-8

Electrical burn Electrical burns on smaller body parts such as the hand frequently result in full-thickness tissue loss with necrosis. The patient with this type of wound should be referred to a regional burn center.

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An electrical burn typically has three injury components that need to be evaluated: a true electrical energy injury from current flow, an arc or flash flame injury by a current arcing at a temperature around 7200°F from the source to the ground, and a flame injury via ignition of clothing. The extent of injury can be easily underestimated because the cutaneous phenotypic appearance may be limited, while underlying muscle necrosis may be extensive. Complications of electrical injuries include rhabdomyolysis leading to hyperkalemia, cardiac injury and arrhythmias, metabolic acidosis, myoglobinemia, and renal failure.

Treatment of electrical burns is focused on aggressive fluid hydration, correction of electrolyte abnormalities, and diuresis. Muscle edema may also lead to compartment syndrome and should be suspected if the patient complains of progressive pain on passive extension and/or paresthesia. In these cases, an emergent fasciotomy is required to prevent progressive tissue hypoperfusion and necrosis. Cardiac arrest, ventricular fibrillation, and cardiac arrhythmias are common within the first 48 hours and can be life-threatening. Indications for cardiac monitoring are documented ECG abnormalities, observed arrhythmias, post-injury electrolyte disturbances, high voltage (≥1,000 V) mechanism, large TBSA burn, and/or advanced age. In addition, transient and permanent nerve injuries, including peripheral neuropathy, delayed transverse myelitis, and anterior spinal syndrome, have been reported.

Lightning strikes are a rare type of electrical injury resulting from a direct current that can generate heat as high as 50,000°F. Due to the short duration of action, usually of 1–2 milliseconds, frank skin necrosis is uncommon. Patients typically demonstrate superficial skin manifestations of a fernlike pattern, termed Lichtenberg figures, that are related to the flow of electrons over the body surface (**FIGURE 10-9**). These lesions are transient, lasting only for 24 hours, and death is usually the result of cardiac arrest and apnea from direct effects of the current on the central nervous system. Lightning can also cause punctate wounds as seen in **FIGURE 10-10**.

FIGURE 10-9

Lichtenberg figures with lacy fern ing pattern seen on the upper chest (Used with permission from Zafren K, Thurman R, Jones ID. Environmental conditions. In: Knoop KJ, Stack LB, Storrow AB, Thurman R, eds. *The Atlas of Emergency Medicine*, 3rd ed. New York, NY: McGraw-Hill; 2010:chap. 16.)

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FIGURE 10-10

Punctate lightning burn (Used with permission from Zafren K, Thurman R, Jones ID. Environmental conditions. In: Knoop KJ, Stack LB, Storrow AB, Thurman R, eds. *The Atlas of Emergency Medicine*, 3rd ed. New York, NY: McGraw-Hill; 2010:chap. 16.)

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Chemical

Chemical burns occur secondary to the contact of the skin with strong acids or bases. Concentration of the agent, quantity, duration of contact, and the mechanism of action in a biological system are factors that determine the severity of the chemical burn injury. Acidic burns tend to be less severe as contact results in a coagulation necrosis of the tissue that creates a denatured protein eschar which then impairs further penetration of the acid. However, basic agents result in a liquefaction necrosis of tissue, which causes a deeper extension of injury due to protein hydrolysis and fat saponification, thereby creating a liquified area of damage that allows the base to penetrate to underlying tissue. Hydrofluoric acid (HF) is unique in that it also causes liquefactive necrosis and penetrates much deeper than other types of acid.

Initial management of chemical burns is focused on removal of the agent from the skin by copious cool water irrigation for extended periods of time, often beyond 30 minutes. The use of warm water should be avoided because it can increase cutaneous porosity and chemical absorption. It is imperative that the irrigation be carried out in the form of lavage instead of submerging the patient in a contained space such as a tub, which will

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prolong skin contact with the caustic source. In addition, neutralization of the acid or base must never be performed as the chemical reaction is exothermic in nature and would cause a thermal injury.

Although neutralization products are usually discouraged, there may be a known specific antidote when the chemical exposure is the result of a single agent. For example, a common acid used in industrial jobs is hydrofluoric acid. The fluoride ion chelates with positively charged ions in the body such as calcium and magnesium, causing an efflux of intracellular calcium and inhibiting biological reactions that require these cations. The fluoride ion will continue to react until neutralized by bivalent cations; thus, calcium gluconate gel or calcium gluconate injections may be used for treatment of these wounds. For severe systemic disturbances of calcium from HF, intravenous calcium gluconate may be given to prevent life-threatening arrhythmias and atony. For most chemical agents, antidotal information is available on the material safety data sheets (MSDS) and should be reviewed to identify potential systemic toxicities and recommended treatment protocols. In addition, regional poison control agencies can be contacted for information on the toxicities of common household products.

Radiation

With the increasing use of ionizing radiation as treatment for disease, more research has focused on understanding the effects of radiation on biological systems. The manifestations of radiation injury are dependent on type of energy source, total dose and time administered, and the location and size of the exposed field. Radiation is most effective on rapidly proliferating cells, making it efficacious for the use in cancer therapy; however, it can also cause “bystander” effects to other normal rapidly proliferating cells in structures such as the skin, gastrointestinal tract, and hematopoietic system.

Radiation-induced skin injury can result in acute and chronic changes to tissues. Acute effects manifest within 6 weeks of radiation exposure as the radiation reacts with water molecules within the cell, causing free radical formation, double-stranded DNA breaks, cell cycle arrest, and apoptosis. In addition to damage to the epithelial and parenchymal cells, radiation induces blood vessel dilation, increased capillary permeability, activation of coagulation, and further necrosis of endothelial cells. Phenotypic changes associated with acute injury include erythema which appears similar to a superficial burn, and higher doses can result in partial-thickness injuries characterized as wet and dry desquamation (**FIGURE 10-11**). Full-thickness ulceration and frank necrosis can result from doses higher than 25 Gray (**FIGURE 10-12**). Acute effects are usually transient and they are amenable to repair by DNA repair mechanisms. After initial exposure to radiation, it takes up to 6 weeks for reversibly damaged epithelial and endothelial cells to recover. Chronic effects, however, can manifest months to years after radiation exposure and are progressive and permanent. The exact mechanism of chronic skin injury is not completely understood; however, there is persistent upregulation of many inflammatory cytokines. One of the most studied pathways in chronic radiation injury is TGF-β and downstream mediator SMAD3 that can signal fibroblasts to produce collagen and lead to extensive dermal fibrosis.[10,11] This is thought to be partly due to sclerosis of small arteries and arterioles, which results in local tissue ischemia and the subsequent release of inflammatory cytokines that promote T-lymphocyte recruitment and further activation of fibroblasts within the injured tissue to thereby create fibrosis.

FIGURE 10-11

Acute radiation burn Changes associated with acute radiation injury include erythema, which appears similar to a superficial burn. Higher doses can result in partial-thickness injuries characterized as wet and dry desquamation, as seen in this patient's face and neck radiation burns.

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FIGURE 10-12

Chronic radiation burn The necrosis, slough, and periwound skin changes including dry desquamation are a result of higher doses of radiation.

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Other phenotypic characteristics of chronic radiation injury are alopecia, pigmentation changes, telangiectasia, impaired vascularity, the development of non-healing wounds, secondary malignancies, and delayed wound healing after insult or surgical procedures (**FIGURE 10-13**). There are limited treatment options for these patients and most wounds have low rates of complete resolution (**FIGURE 10-14**). Many patients will require wide local debridement and autologous tissue coverage. Of recent interest in the literature is the use of lipo-aspirated fat or adipose-derived stem cells for treatment of radiation-induced chronic wounds. Studies have demonstrated that application can lead to improvement and even complete resolution of chronic lesions induced by radiation.[12] Additional research needs to be conducted on the safety and efficacy of its potential therapeutic use, the long-term survival of the adipose tissue, and potential malignant transformation of these multipotent stem cells.

FIGURE 10-13

Chronic radiation wound Chronic radiation wounds rarely heal without surgical intervention. Visible signs of radiation damage include the necrotic bone, periwound skin changes with increased vascularity and sloughing, and loss of hair. Note (**A**) the granulation buds that are coming from the

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trabecular bone under the necrotic cortical layer (in the center) and (**B**) the recurrence of tumor tissue at the edge of the wound.

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FIGURE 10-14

Chronic skin changes after radiation Chronic skin changes after radiation can include pigmentation changes, telangiectasia, and impaired vascularity as seen in this patient who had radiation and reconstructive surgery for breast cancer.

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Contact

Contact burns are the result of skin contact with hot objects including metals, plastic, glass, or coal. The depth of penetration is related to the duration of contact and the temperature of the object. Although these burns are usually small in size, they may have deeper penetration that can result in extensive myonecrosis. One common area is the hand, which can result in a significant functional impairment due to wound contracture if the burn is allowed to heal without any surgical intervention. Treatment is focused on debridement of necrotic tissue and early grafting of areas with deep partial-thickness or full-thickness injuries in order to prevent functional impairments from the development of hypertrophic scars. Evaluation of contact burns in susceptible populations such as children and the elderly must assess for inflicted or non-accidental etiologies when clear lines of demarcation and multiple sites are observed, often with stories that do not match the burn pattern.

Cold

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Thermal injuries from temperatures at the cold end of the spectrum are often overlooked when addressing burn injuries because their geographical dependence often limits presentation to many burn centers. Cold injuries have a slightly different categorization than other thermal injuries and are designated as frostnip (reversible, analogous to first-degree or superficial burns), superficial frostbite (irreversible; partial-thickness injury), and deep frostbite (irreversible; full thickness injury). Cold injuries most commonly occur at temperatures below 28.4°F (−2°C) due to the slow freezing of tissues and subsequent intra- and extra-cellular crystal formation with further exacerbation in the setting of repeated freeze-thaw cycles. Unlike high temperature thermal injuries, low temperature etiologies result in injury that progresses from distal to proximal along extremities and other exposed structures such as the ears and nose. Because the insulting medium is mostly cold air, the injuries are circumferential in nature and rarely spare skin folds or creases as in scald and contact mechanisms.

The factors that influence the extent of local tissues to cold injury are the nadir tissue temperature, duration of exposure, rate of cooling, ischemia time, number of freeze-thaw cycles, and the rewarming conditions. From a pathophysiological perspective, low temperatures result in vasoconstriction of peripheral vascular beds (ie, extremities) to redirect blood from counter-exchange systems that would result in heat loss in order to preserve core temperature. This further exacerbates cooling of the peripheral tissue and additional counter-productive vasoconstriction occurs. At 15°C, maximal vasoconstriction is reached (local blood flow = 20–50 mL/min). To counteract this deleterious cycle, once skin temperatures drop below 15°C, the body undergoes a process called the *hunting response*, defined as cycles of vasodilation and vasoconstriction transiently every 5–10 minutes to facilitate rewarming of extremities and minimize cold-induced tissue damage. The drawback of the hunting response is that it results in delivery of cold blood to the central circulation, cooling the core, and eventually the body must blunt the hunting response to maintain core temperatures. Once skin temperature reaches 10°C, neuropraxia occurs, and below 0°C cutaneous circulation is nearly absent, skin temperature drops 0.5°C/min, and true freezing occurs. Smaller vessels and the venous system are the first to freeze as the increased blood viscosity more easily occludes smaller diameter and lower flow rate vessels.

While rapid freezing, often seen with liquid nitrogen and other industrial accidents, causes intracellular ice crystal formation, the more common environmental exposure cooling is a slow process that starts with extracellular crystal formation. The extracellular crystals create an osmotic pressure gradient that dehydrates cells, creates electrolyte shifts, and induces acid–base disturbances. The ice crystals may further cause direct harm to adjacent cell membranes. Endothelial cells are also injured which, when combined with the increased blood viscosity and activation of clotting cascade from adjacent tissue damage, results in thrombosis of the microvasculature and further tissue ischemia. Studies show that the greatest degree of tissue injury is actually caused by thawing or rewarming of the tissue due to the initiation of reperfusion injury. In particular, restoration of perfusion to the ischemic tissues results in generation of free oxygen radicals, prostaglandin-induced vasoconstriction, leukocyte diapedesis, and clot formation through platelet aggregation.

Superficial frostbite often presents with hyperemia, numbness, and clear or serous blisters (**FIGURE 10-15**). These injuries often are painful on rewarming and sensation is a positive prognostic factor. Deep frostbite results in hemorrhagic blisters due to propagation of tissue injury into the reticular dermis and the underlying subdermal vascular plexus that can further involve muscle and bone (**FIGURE 10-16**). Poor prognostic findings include hemorrhagic blebs proximally distributed on the affected limb in the setting of cold distal tissue that appears ischemic and insensate. Deep frostbite has a firm, woody feel with a blue-gray appearance as opposed to the supple hyperemic character of superficial frostbite. Furthermore, unlike superficial injuries, deeper injuries remain insensate after thawing.

FIGURE 10-15

Superficial frostbite A. Superficial frostbite on the Achilles of an individual who placed a commercial cold pack directly onto the skin with no protective interface material. **B.** The thick blister peeling off with new epithelium beneath. The wound healed with no intervention except time and protection.

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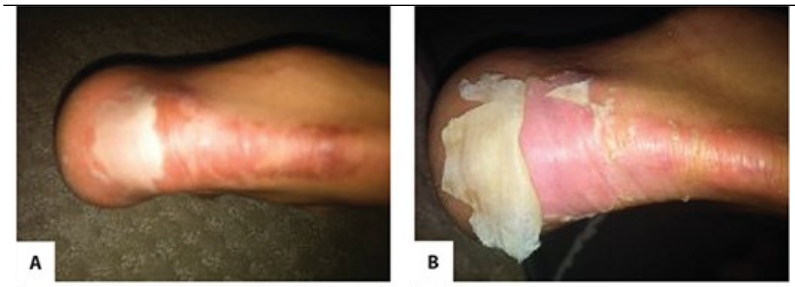

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FIGURE 10-16

Deep frostbite Deep frostbite shows signs of deep tissue injury, edema, erythema, and necrosis. (Used with permission from Dean SM, Satiani B, Abraham WT. Color Atlas and Synopsis of Vascular Diseases. McGraw-Hill, 2014. https://accesssurgery-mhmedical-com.ezproxy.cihe.edu.hk/content.aspx?bookid=1201&sectionid=71015918.)

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Although evaluation and treatment of thermal injuries is addressed in the following section, the unique nature of frostbite treatment will be discussed in this section. The first step in the management of frostbite injuries is to rapidly rewarm the affected extremity for 15–20 minutes with water heated to 38–42°C. Conversion of the frostbitten area to a pliable, soft, and red or purple color indicates the end of rewarming. Numerous studies have looked at the value of different interventions for improving post-rewarming outcomes with varied results. Some studies have shown that amputation rates are decreased with tissue plasminogen activator (tPA) administration within 24 hours of rewarming. The practice of blister deroofing in frostbite is controversial but numerous burn specialists have advocated for aspirating or debriding clear and yellow blisters which classically contain prostaglandin F_{2a} and thromboxane A_2, both of which damage underlying tissue through platelet aggregation and thrombosis. Hemorrhagic blisters are often left intact as these injuries are deeper and often do not benefit from deroofing like the aforementioned blisters. The exposed wound beds from denuded blisters may be dressed with topical antimicrobials in a similar fashion to other burn injuries. Some data exist to specifically support the use of aloe vera, a topical thromboxane inhibitor, on frostbite injuries. Operative interventions in frostbite injury are often deferred at least 10–14 days

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to allow the irreversible cell injury to fully declare itself and the wound to demarcate. Acute surgery is rarely performed but may be done in the case of compartment syndrome associated with severe frostbite injury. Multiple authors including Shenaq and Gottlieb have emphasized the value of triple-phase bone scans in identifying delineations between viable and non-viable bone by their ability to track bone perfusion over the course of the first few days, thereby providing a high predictive value for final amputation level.[13,14]

INITIAL EVALUATION AND MANAGEMENT OF A BURN WOUND

Healing a burn injury depends on appropriate decision making, which begins at the time of initial assessment. The history, including the exact mechanism of injury, and a comprehensive physical examination, including extent of burn, are critical to providing an optimal outcome. The evaluation of the entire patient includes a thorough medical and surgical history, systemic premorbid conditions such as diabetes and cardiovascular disease, current medications, prior trauma to the affected area, and any pre-injury functional deficits. This is critical to planning treatment of the patient with a burn injury. Burns can be relatively small and self-limiting, or can be massively devastating and lead to multi-systemic derangements. Accurate initial evaluation determines the path to success.

Initial Assessment

Initial evaluation of the burned patient should immediately classify the patient as in need of outpatient, inpatient, or critical care in an intensive care unit. This may be rapidly accomplished by including the ABCs of trauma evaluation. Concern for airway stability is evaluated by history and physical examination. Burns that include flame injury or explosions are suspicious for inhalation injury, especially those that occurred in a burning structure or enclosed space. Facial burns, singed facial hair, coughing, hoarseness, voice changes, and stridor may indicate inhalation injury. Physical examination of breathing and ventilatory capacity should be rapidly evaluated with auscultation and observation of a patient's breathing mechanics because progressing tachypnea, labored breathing, or respiratory distress needs to be identified and treated immediately with endotracheal intubation and mechanical ventilation. Even small burns that include an inhalation injury should be triaged to critical care pathways. Following intubation, formal bronchoscopy should be performed to assess whether or not inhalation injury is present and to what extent in order to help guide ventilation and fluid resuscitation management.

Circumferential burn wounds located around the torso can form leathery eschar that compresses and compromises chest expansion, perfusion, and ventilation. Non-circumferential torso injuries that are full-thickness can create a non-compliant eschar that impairs chest excursion and thereby prevent adequate ventilation. As such, it is important to closely examine diaphragmatic dynamics and chest wall mechanics during respiration in the setting of any circumferential or full-thickness injury to the torso. Similarly, circumferential burns around the extremities can constrict perfusion to the extremities through a tourniquet effect, resulting in ischemia and compartment syndrome. Frequent examination of peripheral pulses and neuromuscular exams are critical in identifying patients who may require escharotomies. In these cases, prompt escharotomy (defined as sharp or electrocautery incision through the entirety of the eschar into adjacent unburned tissue) is warranted to separate the constricting band of eschar and relieve compression (**FIGURE 10-17**). Other areas where burn eschar may create restrictive complications in the setting of progressive burn wound edema are around the neck and orbit. Circumferential neck burns require frequent re-assessment of airway patency and evaluation for any signs of neurological decline. Severe facial burns with periorbital eschar can result in orbital compartment syndrome, in which case frequent intraocular pressure assessments are required to identify patients who may require decompressive lateral canthotomies. When assessing burn depth, perhaps the single most important maneuver on the physical exam is palpation of the burn wound. A single maneuver such as dragging a finger across the wound bed with moderate pressure will facilitate assessment of blanching, moisture, and sensation within seconds. Very rapidly, details for determining burn depth may be acquired.

CASE STUDY

INTRODUCTION

Ms RT is a 30-year-old female who sustained a scald injury to abdomen and left thigh when a pot of boiling water accidentally spilled on her while she was cooking. She has no significant past medical history, allergies, or social issues (**FIGURES 10-18** and **10-19**).

DISCUSSION QUESTIONS

1. How is the patient's burn classified?

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2. What would be the total burn surface area, using the Rule of Nines?

Evaluation reveals extensive blistering of the skin, consistent with partial-thickness injury. There are also areas of pale, dry dermis, consistent with either deep partial-thickness or full-thickness injury. The TBSA is determined to be 8% (5% for the abdomen and 3% for the left thigh). The patient is determined to be hemodynamically stable with no substantial fluid loss. She remains alert and oriented ×4, but complains of 8–10/10 pain using the analog scale.

DISCUSSION QUESTIONS

1. What would be the immediate plan of action seconds after sustaining the burn?
2. What would be the best plan of care based on evaluation in the emergency room?
3. What are the major concerns for a patient with scalding wounds during the first 48 hours of treatment?
4. Should this patient be admitted to the intensive care or burn unit?

FIGURE 10-17

Escharotomy of a circumferential burn Circumferential burn wounds located around the torso can form leathery eschar that compresses and compromises chest expansion, perfusion, and ventilation. Similarly, circumferential burns around the extremities can constrict perfusion to the extremities, resulting in compartment syndromes. In these cases prompt escharotomy is warranted to separate the constricting band of eschar to relieve compression.

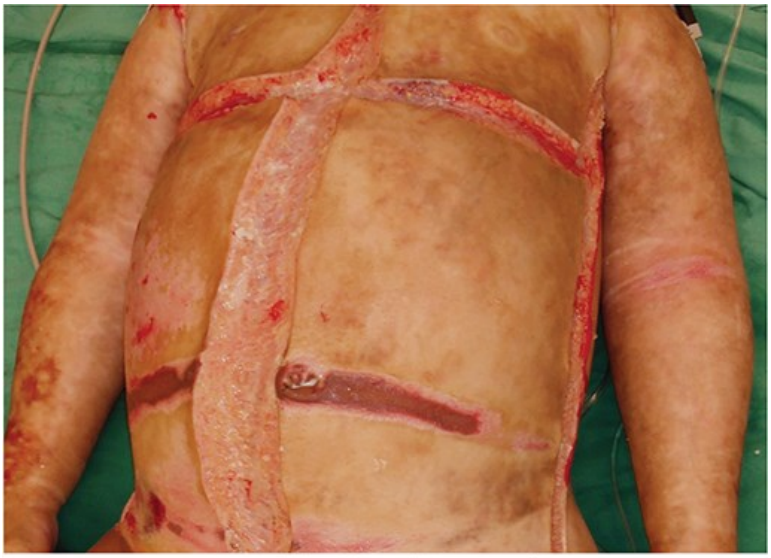

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FIGURE 10-18

Abdominal burn wound

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FIGURE 10-19

Burn wound on the left thigh

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Fluid Requirements

Patients who sustain burns over 20% of their TBSA are at risk of circulatory abnormalities, or burn shock, that compromise perfusion and can lead to end-organ failure and progression of the burn wound to a deeper depth. The goal of fluid resuscitation, therefore, is to maintain adequate end organ perfusion in the setting of burn shock. The pathophysiology of burn shock is not completely understood; however, huge fluid shifts are caused by the leakage of fluid and protein from the intravascular space into the interstitial space. In addition, the release of inflammatory mediators from the burn area creates a hyperdynamic response in extreme cases and can lead to excessive systemic inflammatory responses, with coagulopathy, hemodynamic instability, and organ failure. Patients with critical-sized burns often greater than 20% TBSA require large amounts of fluid resuscitation to prevent organ damage. The Parkland formula is often used to estimate the initial fluid requirement in relation to the body surface area of burn and the patient's weight. The current formula is

$$4\,\text{cc} \times \text{weight in kilograms} \times \%\text{TBSA burn} = \text{24-hour intravenous fluid (IVF) requirement}$$

4 cc×weight in kilograms×%TBSA burn =24-hour intravenous fluid (IVF) requirement

One-half of the calculated fluid requirement is given over the first 8 hours and the last half over the following 16 hours. It is important to note that the target time intervals for resuscitation start from the initial time of burn injury, not the time that intravenous fluids are started. In other words, if a patient is extricated from a car fire and arrives in an emergency room 3 hours after initial injury, the first half of calculated fluids should be delivered within 5 hours as opposed to 8. This formula provides only a rough estimate to guide the initial hourly infusion rate and theoretical 24-hour requirement, thus proper fluid resuscitation must be assessed by closely monitoring the patient's hourly urinary output, which should be maintained at 0.5–1.0 cc/kg/hr, through insertion of an indwelling Foley catheter. Resuscitation infusion rates are then titrated to achieve the desired urine output (UOP) goals and to maintain mean arterial pressures greater than 65 mmHg. Vigilant titration of fluids to the target parameters is important to avoid under and over-resuscitation which both have significant clinical sequelae. Under-resuscitation can lead to burn wound conversion and/or global end-

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organ hypoperfusion, whereas over-resuscitation can cause excessive local and systemic edema that may result in extremity and abdominal compartment syndromes. After the first 8 hours of resuscitation, increasing IVF requirements to maintain UOP goals may indicate a phenomenon known as “fluid creep” (defined as increased third spacing of IVFs) and indicate that initiation of colloid resuscitation may help preserve intravascular volume while reducing overall IVF requirements. If a patient has a burn sufficiently large enough to require fluid resuscitation, intubation, or ICU care, direct management should be provided by a physician trained in burn critical care. The details of management of burns of this extent are beyond the scope of this chapter.

Nutritional Requirements

Large burn injuries can also result in a hypermetabolic state that begins soon after injury and can persist until complete coverage of the wound. Burns greater than 30% TBSA in adults and 20% in children may increase basal metabolic rates by up to twofold, necessitating intensive nutritional supplementation protocols. Even in smaller TBSA burns, the increased caloric requirements are significant, and failure to achieve those needs results in burn wound conversion and delayed healing. Two equations commonly used to predict the caloric requirement are the Curreri formula and the Harris–Benedict formula (**TABLE 10-3**). The Curreri equation has been reported to overestimate and the Harris–Benedict formula to underestimate the exact requirements; thus, an average of the two equations is commonly used. A significant amount of protein is required to prevent protein catabolism and muscle wasting. An estimated requirement of 2 g/kg/day is used by some groups, while others take into consideration the burn surface area (BSA) using the equation of 1 g/kg/day + (3 g × %BSA). Nitrogen balance studies are recommended on a regular basis in order to ensure that sufficient protein intake is maintained. The goal is to start enteral feeding within the first 48 hours to preserve mucosal integrity and prevent bacterial translocation. Insertion of nasogastric tubes (eg, Dobhoff tube) facilitates early initiation of enteral feeding. Previously, post-pyloric positioning was preferred over gastric placement as studies showed decreased rates of feeding intolerance and potentially aspiration events. Furthermore, studies in general surgery and burn populations showed continuous perioperative feeding with post-pyloric placement did not increase aspiration rates and ensured continued caloric supplementation. However, gastric feeding has benefits, such as gastroprotective effects against Curling's ulceration, ability for bolus feeding, and a more physiologic activation of the digestive cascade. Parenteral feeding has been associated with increased infection and mortality rates, so it is frequently avoided unless absolutely indicated, for example, in patients with obstruction, ileus, intestinal discontinuity, abdominal compartment syndrome, or some other malabsorptive pathology.

TABLE 10-3

Formulas for Nutritional Requirements of a Burn Patient

Curreri Formula	
$25\,\text{kcal} \times \text{Weight}\,(kg) + 40\,\text{kcal} \times \%\text{TBSA burn}$	25kcal×Weight(kg)+40kcal×%TBSAburn
Harris–Benedict Equation	
$\frac{\text{Men}}{66.4730} + (13.7516 \times \text{Weight(kg)}) + (5.0033 \times \text{Height(cm)}) - (6.7550 \times \text{Age(yrs)})$	Men66.4730+(13.7516×Weight(kg))+(5.0033×Height(cm))−(6.7550×Age(yrs))
$\frac{\text{Women}}{655.0955} + (9.5634 \times \text{Weight(kg)}) + (1.8496 \times \text{Height(cm)}) - (4.6756 \times \text{Age(yrs)})$	Women655.0955+(9.5634×Weight(kg))+(1.8496×Height(cm))−(4.6756×Age(yrs))

Note: The Curreri formula and Harris–Benedict equation can both be used to calculate the calorie requirements for a patient recovering from burns. The hypermetabolic state, especially in cases of large total burn surface area, requires greater calorie and protein intake in order for the body to synthesize new tissue.

Pain Control

Patients with burns may suffer unrelenting pain that is challenging to control. Current recommendations focus on the management of three aspects of pain: breakthrough, background, and procedural. Goals of pain management are to provide a sufficient amount of pain relief without oversedation in order to allow the burn patient to comfortably engage in dressing changes and participate in rehabilitation exercises.[15] Treatment strategies should be individualized and multimodal in nature, including the use of analgesics, sedatives, and anxiolytics. Utilization of multiple analgesics that alter pain signaling through different pathways (e.g. mu-opioid, arachidonic, Cox-2, GABA, local or regional blocks) or at different sites within the pain pathway is ideal to maximize the total anesthetic effect.[16] Conscious sedation with agents such as fentanyl, ketamine, and propofol is becoming increasingly popular for procedural interventions to provide analgesic and anxiolytic benefits with a potential reduction in procedural trauma.[17,18] Other strategies

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for pain management studied specifically for burned patients include cognitive behavior therapy,[19] music,[20,21] virtual reality,[22] and computer tablet distraction (studied in the pediatric population[23]). Adequate pain control is assessed by monitoring the patient's vital signs, administration of pain scales, and patient report using one of the pain scales discussed in Chapter 3, Examination and Evaluation of the Patient with a Wound. Appropriate pain management is crucial as literature indicates that uncontrolled pain acts as a major physiologic stress that can negatively impact wound healing and patient recovery as well as contributing to post-burn depression, anxiety, and post-traumatic stress disorder.[24]

TREATMENT

Once the initial patient evaluation is completed and life-threatening injuries are assessed and managed, care is directed toward assessing the extent of burn injury and wound treatment. Evaluation of the burn wound is directed at identifying the location, extent, and depth of the burn injury. These characteristics of the burn can then be used to make an assessment of the healing potential of the wound and to determine which interventions will be necessary.

Superficial Burns

Once a superficial burn is diagnosed according to the specifications noted above, treatment is generally supportive. Initial treatment with cool, not cold, compresses can provide relief. Ice packs, while they may provide pain relief, can actually cause burn wounds to convert to deeper injuries due to excessive vasoconstriction and subsequent hypoperfusion of the already injured tissue. These burns can be quite painful, so appropriate analgesia with nonsteroidal anti-inflammatory drugs or codeine may be used as needed. The dermal barrier is not violated, but rather, epidermal desquamation or “peeling” occurs; this can be treated effectively with topical moisturizing lotions. In addition, aloe vera provides both cooling and analgesic effects in superficial burns. Because the barrier function of the skin is intact in a superficial burn, topical antimicrobial treatment is not required. Ultimately long-term contraction and scarring are not encountered, although occasionally transient skin color changes may be noted.

Superficial Partial-Thickness Burns

Burns that disrupt the epidermal barrier are more serious, and must be promptly evaluated and diagnosed. Superficial partial-thickness burns often have large fluid-containing blisters that should be drained and the skin debrided so that the true depth of the wound can be evaluated. These burns, as a result of this epidermal disruption, are at risk for infection and thus should be treated with topical antimicrobial medications. Prophylactic systemic antibiotics can predispose colonization by multidrug-resistant organisms and are, therefore, contraindicated.

Topical Antibiotic Dressings

Silver sulfadiazine (Silvadene) has been the most widely used ointment in the burn population. Silvadene has broad-spectrum antibiotic coverage; however, the silver ion carrier has limited potential for penetrating the burn eschar, making it ideal for superficial and deep partial-thickness injuries. Side effects of Silvadene are thrombocytopenia and neutropenia, and, although, labs are sometimes monitored for these side effects, the neutropenia that develops is not functionally significant and has no impact on infection rates. In addition, this medication should be avoided in patients with sulfonamide allergies, pregnant or nursing women, or infants younger than 2 months.[25] Another downside of Silvadene is the tendency for it to form a pseudoeschar on deeper partial-thickness burns. This pseudoeschar develops when old Silvadene is not completely removed prior to application of new cream, and it creates a thickened, yellowish eschar that complicates burn wound assessment, may be mistaken for pus or infection, and can impair wound healing. As a result, its use in an outpatient setting should be considered in patients where pain control or compliance with daily dressing changes may be a concern.

Mafenide acetate (Sulfamylon) is another topical antimicrobial agent that has broad-spectrum coverage; however, unlike Silvadene it has good penetration of burn eschar and cartilage. One side effect of Sulfamylon is that systemic absorption can cause a metabolic acidosis through inhibition of carbonic anhydrase. Also, Sulfamylon can induce pain upon initial application and thus may limit its use in certain patient populations. As a result, Sulfamylon is often reserved for wounds infected with *Pseudomonas aeruginosa*, deep burns over cartilaginous structures (eg, ears, nose), and burns with thick eschars.

Silver nitrate is another topical antimicrobial agent with broad coverage and unlike Sulfamylon is not reported to induce pain upon application. Side effects include hyponatremia, hypochloremia, and on rare occasions a methemoglobinemia. Silver nitrate is no longer used as frequently as in the past given its suboptimal coverage spectrum, especially in the setting of newer topical antimicrobials and silver-based non-daily dressings. Of the

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commonly used silver delivery dressings, nanocrystalline silver has been shown to be the most potent silver delivery system as compared to hydrofiber and foam dressings that are impregnated with silver and thus more effective in preventing infection, facilitating healing, and shortening length of stay.[26]

Sodium Hypochlorite and Hypochlorous Acid

Classically, Dakin's solution was used as a topical antimicrobial solution deriving its effect from sodium hypochlorite (NaOCl). In order to avoid severe cytotoxicity, it has been progressively diluted down to 0.5% and has consequently lost more than 95% of its antimicrobial effects. Newer products composed of 0.03% hypochlorous acid (H^+OCl^-) have antimicrobial and antifungal effects without the cytotoxicity seen with clinically effective doses of Dakin's solution. As a result, the use of hypochlorous acid as an irrigation agent for skin grafts, debrided wounds, and with other topical enzymatic debriding agents has become an increasingly useful option in the burn wound management arsenal.

Silver-Impregnated Gauze

The use of silver-impregnated gauze dressings has gained favor as they can be used to decrease the frequency of dressing changes (i.e. “non-daily”) and can provide adequate antimicrobial coverage of the burn wounds. These dressings come from a variety of manufacturers, and can be used with or without sterile water, allowing for both the moist wound environment and the elution of the active silver ions to prevent infection. The downside of using these dressings is that in a dynamic deep partial-thickness wound, the non-daily nature of the dressing change frequency delays evaluation for wound progression.

Enzymatic Debridement

For over two decades, various organic extracts from citric fruits and bacterium have been used as enzymatic debriding agents. Enzymatic debriding agents derived from pineapple (ie, bromelain) contain proteolytic and hydrolytic capabilities that result in the decomposition of collagen and other necrotic tissue components in partial-thickness burn wounds. Other enzymatic debriders such as collagenase have been derived from fermenting *Clostridium histolyticum*, and provide a slower yet effective chemical debridement of partial-thickness burn wounds in comparison to the stronger and faster-acting, yet more painful citrus-derived enzymes. Enzymatic debridement is optimally used in mixed partial-thickness burns to facilitate removal of pseudoeschar, coagulum, and other debris over the course of hours to days to allow for more accurate burn depth assessment. Studies[27] have shown that enzymatic topical ointments actually decrease the rate of surgical interventions such as tangential debridement and grafting for mixed partial-thickness burns.

Deep Partial Thickness and Full Thickness

The partial-thickness injury is divided into superficial and deep designations, primarily in order to evaluate the need for surgical intervention. Superficial partial-thickness injuries will likely heal within 2–3 weeks if appropriate antibiotic dressings are applied. As discussed above, the use of enzymatic debridement may decrease the number of superficial and mixed partial-thickness burns that would otherwise require surgical debridement. Deep partial-thickness and full-thickness injuries can take longer to heal and will subsequently lead to more scarring and tissue distortion. Generally, these injuries will not heal within 3 weeks, or have been observed to require more than 3 weeks of conservative management and a grafting procedure. The prolonged inflammatory response that is present in wound beds with protracted closure times stimulates increased fibroblast activity and subsequent collagen deposition and matrix metallopeptidase activity with impaired organization and stabilization of the extracellular matrix. These are two of the main reasons that burn wounds which take longer than 3 weeks to heal have a significantly higher risk (56% vs 8%)[28] for developing hypertrophic scarring and their associated sequelae.

Surgical Treatment

Surgical management is required for full-thickness injuries as well as larger deep partial-thickness injuries that do not show signs of re-epithelialization. The goal of surgical management is removal of nonviable tissue, restoration of the immunologic barrier, and restoration of normal aesthetics as desired in sensitive areas (eg, face, hands, genitalia, breasts, and feet). Tangential excision is employed to remove necrotic tissue in subsequent layers until healthy viable tissue is revealed. Surgical debridement can be challenging because of the large amount of blood loss that is associated with this procedure. Careful excisional procedures and use of intra-lesional epinephrine injections (frequently at dilutions of 1:1,000,000 in

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normal saline) and application of epinephrine-soaked telfa non-adherent gauze can protect against massive blood loss. Excision procedures are performed with the goal of sparing viable tissue while removing nonviable tissue in preparation for coverage with skin grafts from a healthy part of the body.

Autologous skin in the form of skin grafts is the gold standard for permanent wound coverage. Skin grafts can be split-thickness (STSG), which involves the epidermis and part of the dermis, or full-thickness (FTSG), which involves all of the epidermis and dermis. STSGs can be meshed in order to expand for maximal coverage; in addition, meshing promotes better survival because the skin is less prone to the development of fluid collections (eg, hematoma, seroma) beneath the graft (**FIGURE 10-20**). FTSGs are thought to be more aesthetically pleasing as color match, contracture, and scarring are often superior in comparison to burns covered with STSG; however, there is limited donor availability and the harvesting process is labor-intensive and subject to greater failure from hematoma and seroma formation under the grafts. In addition, FTSGs develop less secondary contracture (graft contracture during the course of the healing process). However, due to their limited availability, they are generally reserved for use in secondary reconstruction of functional areas such as the hands or eyelids (**FIGURE 10-21**) where contracture may lead to debilitating functional deficits. Skin can be harvested from unburned parts of the patient's body in a manner that minimizes further disfigurement by hiding the donor sites within “short” and “shirt” boundaries; however, patients who sustain large surface area burns have limited availability of donor sites and therefore any available donor site must be used to achieve coverage. These patients benefit from staged coverage procedures that allow the donor sites to re-epithelialize for later repeat harvest—a process that often takes two or more weeks depending on a patient's healing potential and nutrition optimization.

FIGURE 10-20

Meshed split-thickness skin graft Meshing the donor skin of a STSG enables the skin to cover a larger area and prevents pooling of fluid under the graft.

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FIGURE 10-21

Full-thickness skin graft on a hand

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Biologic dressings can be used as a temporizing method of wound coverage to prevent further fluid and heat loss and to lower the rates of wound infection. The most commonly used biologic dressing is *allograft* (human cadaveric split-thickness skin). When placed on a healthy wound bed it promotes revascularization; however, it will undergo rejection within 1–2 weeks as the newly formed capillary beds facilitate delivery of immune cells to the allograft's dermis. A potential disadvantage is transmission of communicable diseases from the donor to the patient, in particular the risk of cytomegalovirus. *Xenografts* (split thickness from another species, commonly porcine) are another option for temporary coverage; however, they have suboptimal adherence, higher incidence of infections, and poor revascularization. Integra® (Integra LifeSciences, Plainsboro, NJ) is a dermal matrix composed of bovine collagen cross-linked with shark chondroitin 6-sulfate glycosaminoglycan and an outer silicone dual-layer membrane. It serves as a scaffold for ingrowth of fibroblasts and blood vessels, and the silicone membrane acts as a temporary barrier (**FIGURE 10-22**). Engraftment is complete in 14–21 days and as vascularization occurs within the matrix, the outer silicone membrane is removed to reveal the "neo-dermis," and is covered by a thin autologous skin graft. Integra is a valuable product when poorly vascularized structures such as bone without periosteum, tendon without peritenon, and neurovascular bundles are in the base of the wound bed and direct application of a skin graft would result in either graft failure or undesired adherence to underlying critical structures. Other cellular and tissue biologic dressings that have been reported as beneficial in treating burns include Apligraf (Organogenesis, Canton, MA), dehydrated Human Amnion/Chorion Membrane (dHACM) allografts (EpiFix, AmnioFix, EpiBurn; MiMedx Group Inc., Marietta, GA[29]), and MatriStem® burn matrix (ACell, Columbia, MD).

FIGURE 10-22

Biological skin equivalent dressings Integra is a dermal matrix composed of bovine collagen cross-linked with glycosaminoglycan and an outer silicone membrane. It serves as a scaffold for in-growth of fibroblasts and blood vessels and the silicone membrane acts as a temporary barrier. These biological dressings are termed living skin equivalents.

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The function of any of the cellular and tissue biologics is to promote wound closure through increased cellular migration and synthesis of extracellular matrix.

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COMPLICATIONS

Hypertrophic scarring is the result of wounds that heal with an excessive amount of collagen formation. Unlike keloids, they do not extend beyond the area of the initial wound, and thus appear as a raised scar within defined boundaries (**FIGURE 10-23**). Ethnic populations with Fitzpatrick types 4 and above (darker skin) are at increased risk of both hypertrophic scarring and keloid formation. Hypertrophic scars develop when deep partial-thickness or full-thickness injuries are allowed to heal by contracture, not re-epithelialization, and in functional areas can lead to contractures and joint impairment (**FIGURES 10-24** and **10-25**). Treatment of hypertrophic scars includes pressure therapy with custom-made compression garments, corticosteroid injections, silicone gel dressings, therapeutic scar massage, and laser therapy, all with varying degrees of scar improvement. In the event of wound contracture causing functional impairment, surgical release, local tissue rearrangement, skin grafting, and/or free vascularized tissue transfer may be warranted (**FIGURES 10-26** and **10-27**).

FIGURE 10-23

Keloid scarring (Used with permission from Keloids. In: Usatine RP, Smith MA, Chumley HS, Mayeaux EJ, Jr. eds. *The Color Atlas of Family Medicine,* 2e, New York, NY: McGraw-Hill; 2013:chap. 204. Available at https://accessmedicine-mhmedical-com.ezproxy.cihe.edu.hk/content.aspx?bookid=685&Sectionid=45361278.)

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FIGURE 10-24

Hypertrophic scarring

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FIGURE 10-25

Hypertrophic scarring with joint impairment at the shoulder

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FIGURE 10-26

Hypertrophic scarring with severe contractures and functional impairments of the hand

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FIGURE 10-27

Surgical excision of hypertrophic scarring may be required for both function and aesthetics

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Burn wound infections, cellulitis, and burn sepsis are currently the greatest contributors to burn morbidity and mortality in patients admitted for burn treatment, accounting for over 75% of in-hospital post-burn complications.[30] Burn infection is diagnosed by a combination of clinical exam, laboratory tests, and quantitative tissue biopsy to identify the causative organism(s) for directing antimicrobial and surgical interventions. These complications are addressed aggressively as wound sepsis is the primary cause of mortality in patients with large burn injuries.

Curling ulcers can occur in the gastric or duodenal mucosa of the gastrointestinal tract of severely burned patients as a result of reduced plasma volume, decreased tissue perfusion, and subsequent gastric mucosal ischemia and necrosis. If allowed to progress, these ulcers can result in hemorrhage and perforation. The incidence of curling ulcers has significantly decreased with the initiation of prophylactic treatment with H_2 blockers, proton pump inhibitors, and early enteral gastric feeding. These ulcers previously contributed to significant morbidity and even mortality in burn patients, and thus prophylactic treatment should be initiated as early as possible if gastric enteral feeding is not initiated early on in treatment.

Burn wounds can result in chronic inflammation that predisposes the area to malignant transformation termed Marjolin ulcers, aggressive ulcerating squamous cell carcinomas, which can occur decades after the inciting event. Marjolin ulcer is suspected if there is a chronic, slow-growing lesion in the area of an old burn wound that classically progresses beyond the boundaries of the initial burn wound. Diagnosis is made by wedge resection; however, these cancers tend to be aggressive and are associated with a poor prognosis. Wide local excision with at least 1 cm margins is required to provide some survival benefit in this population.

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Heterotrophic ossification (HO) is a rare complication related to full-thickness burns near joints due to elevated TGF-β cross-reactivity with bone morphogenic protein (BMP) pathways present in nearby periosteum. HO usually occurs 1–3 months postinjury and has a higher incidence in the upper extremities, in particular, at the elbows. Calcium is deposited in soft tissues surrounding the involved joint, thus leading to impaired movement as heterotrophic bone is formed within the soft tissue. Plain films demonstrating calcification of the soft tissue and muscle (myositis ossificans) around the involved joint can be used to confirm diagnosis. Treatment in many cases involves excision of the deposits in order to restore range of motion; unfortunately, however, rates of recurrence may be as high as 35% even after surgical resection and early initiation of postoperative therapy.

REHABILITATION

Early rehabilitation of the burn patient focuses on range of motion exercises and splinting to prevent wound contracture and functional limitations. Long-term rehabilitation places greater emphasis on restoration of activities of daily living, correction of cosmetic deformities, and physiological adaptation. Occupational and physical therapists play an integral role in the treatment plan even in the initial stages of burn treatment. Many of these injuries are life-changing experiences, leading to dramatic physical adjustments that cause severe psychological stress. Newer strategies of burn rehabilitation integrate psychological assessment to identify and treat early signs of posttraumatic stress disorders and/or depression. The incorporation of virtual reality paradigms to simulate important daily tasks as well as to motivate pediatric patients has become an area of increasing interest and success. This integral approach of physical and occupational therapy involvement from initial hospital admission through long-term outpatient follow-up aims to address all levels of recovery with the ultimate goal of returning the patient to the previous level of function.

CASE STUDY

CONCLUSION

The patient arrived at the emergency room with clothes still intact, so they were carefully removed to avoid further skin damage. Because of the pain levels, intravenous narcotic medication was administered to initially control pain. The burns were irrigated and debrided of devitalized tissue, once adequate pain control has been established. Because the wounds were partial thickness, a topical antibiotic dressing was applied and no systemic antibiotics were indicated since the patient had no other comorbidities. Classic choices include topical silver dressings, such as silver sulfadiazine, which are inexpensive but laborious, requiring daily dressing changes. In addition, they require thorough irrigation to prevent buildup of residue, which can be painful to remove and inhibit evaluation of the burn. Other options include silver impregnated gauze that can be placed after initial debridement and left for up to 7 days. These dressings are cost effective, and prevent the pain associated with daily dressing changes. They are effective at preventing infection. Also, because it was too early to determine if the wounds were superficial or deep partial thickness, the patient was admitted for daily care until it could be determined if further surgical debridement of any devitalized tissue would be needed. If the injuries only result in superficial partial-thickness injuries, re-epithelialization should occur with normal wound healing without any additional intervention provided there is no infection. However, if the patient has deep partial-thickness injuries, early surgical debridement and grafting is warranted to prevent future hypertrophic scarring and wound contracture.

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